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UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

NEETA THAKUR, et al.,

Plaintiffs,

v.

DONALD J. TRUMP, et al.,

Defendants.

Case No. 3:25-cv-4737

DECLARATION OF ALEXANDER VAN DER BLIEK

DECLARATION OF ALEXANDER VAN DER BLIEK

I, Alexander van der Bliek, declare as follows:

1. I have personal knowledge of the facts contained in this declaration and, if called as a witness, could and would testify competently to those facts.

2. I am a Professor of Biological Chemistry at the University of California Los Angeles (UCLA), where I have been employed for thirty-two years.

3. I earned my PhD at the Netherlands Cancer Institute in the Netherlands in 1988. From 1988-92, I conducted postdoctoral research at CalTech. In 1993, I was hired at UCLA as an Assistant Professor of Biological Chemistry; in 2000, I was promoted to Associate Professor; and in 2006, I was promoted to full Professor.

4. My research honors include fellowships with EMBO (an international membership organization promoting excellence in the life sciences) and HFSPO (the Human Frontiers of Science Organization), and a five-year role as Research Scholar at the American Cancer Society. I also served as member of a grant review panel for the American Heart Association, Western States Division, for six years. During the past two decades I have additionally served as a regular member and a temporary member of multiple NIH study sections. Study sections are groups of 20-25 scientists who review grant applications for the NIH from others in the same field and determine the scores that will help decide who gets funded. This is a crucial aspect of the NIH granting system, as it ensures that limited resources are allocated to the best, most promising, and most feasible projects.

5. My laboratory studies the role of mitochondria—often referred to as the powerhouses of the cell—in neurodegenerative diseases. These diseases include Alzheimer's and peripheral neuropathies (diseases that damage nerves outside the brain and spinal cord, causing chronic pain, tingling, and other symptoms). My lab uncovered the molecular basis of mitochondrial fission, which is essential for cell survival but also plays a critical role in cell death. These findings helped establish mitochondrial dynamics as a central area of cell biology. Today, this vibrant field is recognized not only for its relevance to neurodegenerative diseases but also for its broad impact on conditions with high energy demands, including cancer and diabetes.

6. I have authored more than 75 peer-reviewed publications in scientific journals. A complete list is in my National Library of Medicine bibliography: https://www.ncbi.nlm.nih.gov/myncbi/alexander.van%20der%20bliek.1/bibliography/public/. These publications have been cited more than 22,000 times: https://scholar.google.com/citations?user=AaU_YIcAAAAJ&hl=en.

7. Over the course of many years at UCLA, I have had multiple cycles of four- or five-year NIH grants, as well as grants from non-governmental agencies, which have dependably funded our research on mitochondrial dynamics. The August 2, 2025 notice I received from UCLA informing me of NIH's suspension of my current, already-awarded multi-year RO1 grant is the first grant disruption I have experienced in any context.

8. A true and correct copy of my biographical sketch is attached as Exhibit A.

Application for Grant Funding from NIH

9. On April 29, 2020, I submitted, in conjunction with the UCLA Office of Contract & Grant Administration, an Application for Federal Assistance to the NIH's National Institute of Neurological Disorders and Stroke (NINDS) for a project titled "Control of Calcium Flux and Mitochondrial Fission by the Charcot Marie Tooth Disease Protein Mfn2" (the "R01 Application"). RO1 grants are large, multi-year, highly competitive funding awards that enable research teams to sustain an extended research program and make corresponding significant contributions to scientific knowledge.

10. The Project Summary in the grant application describes our research into the underlying causes of Charcot-Marie-Tooth (CMT) disease. Charcot-Marie-Tooth disease (CMT) is an inherited condition that damages the nerves controlling movement and sensation. In our study, we looked at how mutations in a protein called Mfn2 affect the way mitochondria, the cell's "power plants," divide. Because this process is closely tied to nerve cell health, our work may help explain what causes CMT and point toward new ways to treat it.

11. The R01 Application requested $2,243,240 for a five-year period (1/1/2021 - 12/31/2025). I was identified as the Project Director and Principal Investigator on the Application. The proposal would fund salaries for myself, one lab technician, and two

postdoctoral researchers, as well as supplies and research costs, travel, and publishing.

12. The RO1 Application is attached as Exhibit B.

Award of Grant Funding for R01 Grant

13. My Application was funded in the amount proposed across all years. The NIH Notice of Award is attached as Exhibit C.

14. I received my most recent Notice of Award Action from UCLA grant administrators on January 10, 2025, informing me that I was to receive the last installment of the grant ($342,488) to sustain the project's final year (calendar year 2025). This Notice is attached as Exhibit D.

Suspension of Grant Funding

15. On August 1, 2025, I received from UCLA's research administrators a "Stop Work Notice" for my active NIH grant. The communication explained that NIH-NINDS had issued a "suspension notice" to UCLA that encompassed my project. I was instructed to immediately stop incurring expenses against the grant award. I was also told to submit to the sponsoring agency a report of expenditures through July 31, 2025, as if this were the closeout of a discontinued grant.

16. I was not offered any reason for the suspension of my grant; any means of appealing this suspension; or informed of any other action I could take to reinstate the grant.

17. A true and correct copy of the Stop Work Notice is attached as Exhibit E.

Harm Suffered from Termination of Grant Funding

18. I and my project team have suffered immediate harms as a result of NIH's actions in suspending this grant. These harms are continuing. Specifically:

19. My lab has one technician and two postdoctoral researchers. My only source of funding is the NIH grant that was suspended. As a result, we are no longer able to purchase supplies for our experiments. I will soon have to let my staff go for lack of University support.

20. The disruptions caused by layoffs are twofold. First, research in my lab will grind to a halt. Any temporary lull, even for a few months, has lasting consequences in terms of my research productivity, our laboratory's output, and the publications I produce with postdocs as co-authors.

Related: It took a year and a half to identify the current postdocs who now work with me as collaborators, to wait for them to complete their PhD training, and to bring them into the lab. If I had to repeat this process now, I would be forced to close the lab due to the considerable delay. That would be a real shame, as a lot of exciting new data would be lost.

21. Second, the postdocs will be unable to complete their projects and publish associated papers. These postdocs are highly specialized, having trained for years in this area, and should still have their careers ahead of them. A gap in publications resulting from layoffs will make them far less desirable in the job market and potentially make them unemployable. Thus, the time I have dedicated to their professional mentorship will have mostly been wasted, and the field as a whole will suffer from the loss of these promising researchers. This will have an enduring adverse impact on research in my subfield of biological chemistry.

22. The U.S. public, which ultimately funds NIH grants, will also lose much of the value of their investment if my NIH grant is indefinitely suspended. Thus far, the work supported by this grant has yielded new insights into the root causes of hereditary neuropathies, opening promising directions for future therapies for these often debilitating disorders. Unexpectedly, the research has also revealed toxic cellular mechanisms linked to brain diseases such as Alzheimer's and frontotemporal dementia. With the opportunity to complete the planned studies, our team aims to share these findings through publications and pursue follow-up experiments aimed at developing treatments. In turn, these publications will help advance public understanding of neurodegenerative and metabolic diseases, including Parkinson's, Alzheimer's, diabetes, and cancer.

Executed this 19th day of August 2025, in Los Angeles, California.

Alexander van der Bliek